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ADHD Comorbidity in Tourette Syndrome: Associations with Tic Severity, Functional Burden, and Clinical Management - A Focused Narrative Review

Dhairya***Student, NIMS Universit, Jaipur, Rajasthan, India***Corresponding Author Email:** Dhairya7484@gmail.com**ABSTRACT**

Tourette syndrome (TS) is a neuro developmental disorder characterized by persistent motor and vocal tics and frequently accompanied by psychiatric comorbidities. Attention-deficit/hyperactivity disorder (ADHD) is among the most common co-occurring conditions and may contribute substantially to the clinical burden experienced by individuals with TS. However, the relationship between ADHD and tic severity is not straightforward, and functional impairment cannot be inferred from tic severity alone. This focused narrative review examines evidence concerning the association between ADHD comorbidity and tic severity, functional and behavioral burden, and treatment response in individuals with TS. Evidence from symptom-network research suggests substantial interaction between ADHD and TS symptoms, while clinical studies provide mixed findings regarding whether ADHD is independently associated with greater tic severity. In contrast, ADHD symptoms appear more consistently related to quality-of-life difficulties, behavioral dysregulation, and broader functional burden. Pharmacological evidence indicates that ADHD can be treated in children with TS without necessarily worsening tics, with α 2-adrenergic agonists and combined stimulant- α 2-agonist treatment showing benefits across ADHD and tic outcomes in available trials. Behavioral interventions, including Comprehensive Behavioral Intervention for Tics (CBIT), also appear effective regardless of ADHD comorbidity. Mechanistic findings involving performance monitoring, genetics, and endocannabinoid signaling provide possible explanations for overlapping vulnerability but remain insufficient to establish causal pathways in humans. Overall, ADHD in TS should be conceptualized as an important contributor to clinical burden rather than as a simple determinant of tic severity. Future research should use longitudinal and multimodal designs to

distinguish shared vulnerability, symptom interaction, and causal mechanisms.

Keywords: *Tourette syndrome, attention-deficit/hyperactivity disorder, ADHD, tic severity, functional impairment, quality of life, CBIT, comorbidity.*

1. Introduction

Tourette syndrome (TS) is a neurodevelopmental disorder characterized by persistent motor and vocal tics. Although tics are the defining clinical feature of TS, the disorder frequently occurs alongside other psychiatric and behavioral difficulties. Attention-deficit/hyperactivity disorder (ADHD) is one of the most frequently reported comorbidities and can introduce additional difficulties involving attention, impulsivity, behavioral regulation, academic functioning, and social adjustment.

The presence of ADHD in individuals with TS raises an important clinical question: does ADHD contribute to greater tic severity, or does it primarily increase the broader functional burden associated with the disorder? This distinction is clinically relevant because tic severity and overall impairment are related but not interchangeable constructs. A patient may experience relatively mild tics while still having substantial difficulties because of inattention, impulsivity, obsessive-compulsive symptoms, emotional dysregulation, or social consequences.

Existing literature provides inconsistent evidence regarding the relationship between ADHD and tic severity. Some studies suggest that greater ADHD symptom severity is associated with greater perceived tic worsening, whereas other clinical samples have found no significant difference in tic severity between individuals with and without ADHD. At the same time, evidence concerning quality of life and behavioral burden suggests that ADHD may contribute meaningfully to impairment even when its association with tic severity is weak or inconsistent.

Recent research has also begun to examine TS and ADHD as interacting symptom systems rather than completely independent disorders. Symptom-network analyses identify connections between attentional, behavioral, and tic-related symptoms, while electrophysiological, genetic, and preclinical research suggests partially overlapping neurobiological mechanisms.

The present focused narrative review therefore examines four related questions: (1) whether ADHD comorbidity is associated with greater tic severity in TS; (2) whether ADHD contributes to functional and behavioral burden independently of tic severity; (3) how ADHD comorbidity influences treatment considerations and outcomes; and (4) what current mechanistic evidence suggests about shared vulnerability between ADHD and TS.

2. Methods

2.1 Review design

This study was conducted as a focused narrative review rather than a systematic review or meta-analysis. The purpose was to integrate clinically relevant evidence concerning ADHD comorbidity in TS across tic severity, functional burden, behavioral symptoms, treatment, and possible mechanisms.

2.2 Search strategy

Literature was identified through searches of PubMed/MEDLINE, Google Scholar, and publisher or journal databases. The primary search concepts combined terms relating to TS, ADHD, tic severity, functional impairment, quality of life, treatment, and mechanisms.

The principal search string was:

("Tourette syndrome" OR "Tourette disorder" OR "tic disorder") AND ("ADHD" OR "attention-deficit/hyperactivity disorder") AND ("tic severity" OR "quality of life" OR "functional impairment" OR "rage attacks" OR treatment OR CBIT OR comorbidity)

Additional focused searches were conducted for pharmacological treatment, behavioral therapy, symptom-network analysis, electrophysiological findings, genetic evidence, and preclinical mechanisms.

The main contemporary search was conducted in September 2026. Earlier studies were also retained when they provided direct foundational evidence relevant to functional burden or treatment and were necessary to address gaps identified in the review question.

2.3 Selection and synthesis

Titles and abstracts were reviewed for relevance, followed by full-text assessment where available. Reference lists of relevant studies were also examined to identify additional directly relevant evidence.

Studies were retained when they addressed at least one of the following domains: ADHD–tic severity relationships, quality of life or functional burden, behavioral dysregulation, ADHD treatment in TS, behavioral treatment for tics in the presence of ADHD, or possible shared mechanisms.

Because this was a focused narrative review and the original screening process was not prospectively logged, an exact retrospective count of records identified, screened, and excluded could not be reliably reconstructed. No fabricated screening numbers are therefore reported. The synthesis emphasizes direct clinical evidence and uses mechanistic studies primarily to provide explanatory context rather than causal conclusions.

3. ADHD and TS as an Interconnected Symptom System

ADHD and TS are clinically distinct disorders, but their symptoms can interact in ways that make the overall presentation more complex. Attention difficulties, impulsivity, motor and vocal tics, compulsive behaviors, and emotional dysregulation may occur simultaneously and influence one another.

A symptom-network perspective is useful because it does not require ADHD to directly cause tics. Instead, it allows individual symptoms to be examined as interconnected components of a broader clinical system.

3.1 Evidence from symptom-network analysis

Zhang et al. (2024) examined TS and ADHD symptoms in a large sample of 3,958 children and adolescents aged 6–16 years. Their network contained 38 symptom nodes, with 18 ADHD symptoms and 20 TS-related symptoms. The authors reported 1,444 potential edges using their network-estimation convention and identified 640 non-zero edges across the full network.

For cross-domain relationships, the 20 TS symptoms and 18 ADHD symptoms produced 360 possible ADHD–TS connections. Of these, 83 were non-zero, corresponding to 23.06% of possible cross-domain connections. The authors also reported 36 non-zero ADHD–ADHD connections out of 80 possible connections and 45 non-zero connections between tic and obsessive-compulsive symptom dimensions out of 64 possible connections.

The study identified difficulty sustaining attention during tasks or play as the most central symptom in the network. Several symptoms also functioned as bridges between symptom domains, including difficulties controlling movements, repetitive movements, uncontrollable words, and excessive talking.

These findings support the view that ADHD and TS symptoms can form an interconnected clinical system. However, network centrality does not demonstrate causation. Because the study was cross-sectional, it cannot determine whether ADHD symptoms contribute to tic symptoms, tic symptoms increase attentional difficulties, or both arise partly from shared developmental vulnerability.

4. ADHD and Tic Severity

4.1 Evidence suggesting an association

Araújo et al. (2022) examined 22 individuals with TS and comorbid ADHD and/or obsessive-compulsive disorder during the COVID-19 pandemic. Participants ranged from 16 to 54 years of age. More than half of the participants reported that their tics had become a little or much worse during the pandemic.

Importantly, ADHD symptom severity was positively associated with perceived tic worsening, with a substantial correlation between the measures ($r = .602$, $p = .003$, 95% CI [.24, .82]). Participants who reported tic worsening also had significantly higher ADHD symptom scores than those who did not report worsening ($Z = -2.376$, $p = .017$).

These findings suggest that ADHD symptom severity may be associated with perceived changes in tic severity under conditions of increased environmental stress. However, the study was small, cross-sectional, and based substantially on self-report. The pandemic context also introduces potential effects of stress, disrupted routines, social isolation, and treatment changes. Therefore, the findings should be interpreted as evidence of association rather than evidence that ADHD causes more severe tics.

4.2 Evidence that ADHD does not necessarily increase tic severity

Evidence from other clinical samples challenges a simple ADHD-to-tic-severity model.

Pringsheim (2017) examined 114 children with TS and found no significant difference in Yale Global Tic Severity Scale scores between children with and without ADHD. However, children with ADHD were more likely to have received treatment for tics during the first two years following presentation, with an odds ratio of 3.51 ($p = .009$).

This finding is important because it separates tic severity from treatment need. ADHD may increase the clinical complexity of TS and influence clinicians' decisions to initiate treatment even when objective tic severity is not substantially different.

Taken together, the available evidence does not support the conclusion that ADHD consistently produces more severe tics. Instead, ADHD may influence how tics are experienced, managed, or functionally disruptive in particular contexts.

4.3 Interpreting the ADHD–tic relationship

The inconsistent relationship between ADHD and tic severity may reflect several factors. First, ADHD and tic symptoms can fluctuate independently. Second, environmental stressors may simultaneously worsen attention, behavioral regulation, and tic expression. Third, comorbid obsessive-compulsive symptoms and anxiety may influence both tic severity and perceived impairment.

Consequently, ADHD should not be conceptualized as a direct and universal determinant of tic severity. A more appropriate interpretation is that ADHD may interact with tic symptoms within a broader clinical system and may be particularly important for understanding treatment needs and functional outcomes.

5. ADHD and Functional and Behavioral Burden

5.1 Quality of life

Quality of life is an important outcome because tic severity alone does not capture the full impact of TS.

Bernard et al. (2009) examined 56 children with TS and investigated associations among tic severity, ADHD symptoms, obsessive-compulsive behaviors, and quality of life. Their findings indicated that motor and phonic tic severity were not significantly associated with quality of life, whereas ADHD and obsessive-compulsive symptoms were related to poorer quality of life. In particular, inattentiveness was an important predictor of lower quality of life.

This distinction is clinically important. A child may have relatively manageable tics but still experience significant academic, social, or emotional difficulties because of inattention and other comorbid symptoms. Therefore, improvement in tic severity should not automatically be interpreted as equivalent to improvement in overall functioning.

The available evidence consequently supports treating functional burden as a separate clinical domain rather than simply assuming that greater tic severity produces greater impairment.

5.2 Behavioral dysregulation and rage attacks

Behavioral dysregulation represents another domain in which ADHD may be clinically relevant.

Cintin et al. (2026) examined rage attacks in a longitudinal and cross-sectional sample of 314 individuals with TS aged 5–19 years at baseline. Participants with rage attacks had higher ADHD symptom scores and more compulsive symptoms. In longitudinal analyses, higher baseline ADHD symptoms were also associated with the presence of rage attacks at follow-up.

These findings suggest that ADHD symptoms may contribute to behavioral dysregulation in TS. However, rage attacks are likely to reflect multiple interacting factors, including tic severity, obsessive-compulsive symptoms, emotional regulation difficulties, and developmental factors. ADHD should therefore be viewed as one contributor rather than a sole explanation.

5.3 Healthcare burden and access

Functional burden may also influence the need for healthcare services. Karacuschansky et al. (2025) examined telehealth use and healthcare delivery among Medicaid beneficiaries with ADHD and TS. Their work primarily addresses access to and quality of healthcare rather than direct measurement of functional impairment.

This distinction is important. Healthcare utilization and access are indicators of clinical burden or service needs, but they should not be presented as direct measures of functional impairment. Telehealth may provide an important means of improving access to specialized care for individuals with complex neurodevelopmental presentations, but the study does not establish that ADHD itself causes greater functional impairment.

6. Behavioral and Pharmacological Treatment

6.1 Pharmacological treatment of ADHD

Treatment of ADHD in individuals with TS has historically raised concern about potential tic exacerbation, particularly with stimulant medications. Contemporary evidence does not support a blanket assumption that stimulants worsen tics.

Farhat et al. (2024) conducted a systematic review and network meta-analysis of eight double-blind randomized controlled trials involving 575 participants with ADHD and TS or tic disorders.

Compared with placebo, α 2-adrenergic agonists produced improvements in ADHD symptoms (SMD = -0.72 , 95% CI [-1.13 , -0.31]) and tic severity (SMD = -0.70 , 95% CI [-0.96 , -0.45]). Stimulant treatment improved ADHD symptoms (SMD = -0.54 , 95% CI [-1.05 , -0.03]), while its effect on tic severity was not statistically significant (SMD = -0.22 , 95% CI [-0.49 , 0.05]). Combined stimulant and α 2-agonist treatment also improved ADHD symptoms (SMD = -0.84 , 95% CI [-1.54 , -0.13]) and tic severity (SMD = -0.60 , 95% CI [-1.04 , -0.17]).

However, the certainty of evidence was rated low or very low for several comparisons. These findings therefore support individualized treatment rather than a universal medication recommendation.

6.2 Behavioral treatment

Behavioral interventions are another important component of treatment for TS.

Sukhodolsky et al. (2017) examined predictors and moderators of response to behavioral therapy for tics using data from two multisite studies involving a combined sample of 248 participants. Comprehensive Behavioral Intervention for Tics (CBIT) reduced tic severity compared with psychoeducation and supportive therapy. Importantly, co-occurring ADHD, obsessive-compulsive symptoms, and anxiety did not significantly moderate response to CBIT.

Similarly, Mazur-Lainé et al. (2024) examined 55 individuals with TS who completed a 14-week cognitive-behavioral intervention for tics. ADHD symptomatology did not significantly alter treatment-related improvement immediately after treatment or at six-month follow-up.

These findings suggest that ADHD comorbidity should not automatically be considered a barrier to behavioral treatment for tics.

6.3 Overall treatment interpretation

The treatment literature supports a multidimensional approach to TS with ADHD. Tic-focused treatment and ADHD-focused treatment should not be viewed as mutually exclusive. Instead, treatment planning should consider which symptom domains produce the greatest functional burden.

The available evidence also indicates that concerns about inevitable tic worsening from stimulant treatment may be overstated. Medication selection should nevertheless remain individualized, particularly because the available evidence base is limited and certainty is often low.

7. Mechanistic and Biological Perspectives

7.1 Electrophysiological evidence

Performance monitoring has been implicated in both TS and ADHD, although the direction and nature of abnormalities may differ.

Bellato et al. (2021) conducted a systematic review and meta-analysis covering 97 studies and 5,890 participants across OCD, GTS, ADHD, and autism. The meta-analysis found increased error-related negativity (ERN) in GTS and OCD but reduced ERN in ADHD and autism. These findings suggest partially overlapping but not identical alterations in performance-monitoring systems across neurodevelopmental conditions.

For the present review, these findings provide mechanistic context rather than evidence that ADHD causes tic symptoms. Differences in performance monitoring may represent one pathway through which ADHD and TS overlap while still producing distinct behavioral phenotypes.

7.2 Genetic evidence

Wang et al. (2023) investigated rare X-linked genetic variants in autism, TS, and ADHD. Their findings identified predominantly male-associated risk patterns across these neurodevelopmental conditions and provided evidence for partially shared genetic vulnerability.

Such findings support the possibility that TS and ADHD may share aspects of their developmental liability. However, genetic overlap does not establish that ADHD symptoms cause tic symptoms. Instead, both conditions may emerge partly from shared biological risk factors.

7.3 Preclinical evidence

Gorberg et al. (2025) used a mouse model to investigate the consequences of postnatal disruption of cannabinoid receptor signaling. Disruption of CB1 and CB2 receptor signaling produced behavioral features resembling aspects of TS and ADHD.

This study provides a possible mechanistic pathway linking the two phenotypes. However, because the findings are based on an animal model, they should be interpreted as preclinical evidence. They cannot establish that the same mechanism explains ADHD–TS comorbidity in humans.

7.4 Integrating mechanistic evidence

Taken together, electrophysiological, genetic, and preclinical findings suggest that TS and ADHD may share partially overlapping developmental mechanisms. At the same time, these mechanisms are unlikely to provide a single explanation for the clinical relationship between the disorders.

The mechanistic literature therefore supports a model of shared vulnerability and interacting symptom systems rather than a simple causal pathway from ADHD to tic severity.

8. Clinical Implications

The clinical significance of ADHD comorbidity in TS extends beyond tic severity.

First, assessment should include ADHD symptoms even when tic severity appears relatively modest. Inattention and impulsivity may contribute substantially to academic, social, and everyday difficulties.

Second, clinicians should distinguish tic severity from functional impairment. A reduction in tics is clinically valuable but may not produce equivalent improvement in quality of life if ADHD, obsessive-compulsive symptoms, anxiety, or emotional dysregulation remain untreated.

Third, treatment decisions should be based on the individual's most impairing symptom domains. Current evidence does not justify avoiding stimulant treatment solely because a patient has TS, although monitoring remains appropriate.

Finally, behavioral treatment such as CBIT should remain part of the treatment discussion because available evidence suggests that ADHD comorbidity does not prevent meaningful response to tic-focused behavioral interventions.

9. Integrating the Evidence

Across the reviewed literature, three conclusions emerge.

First, the association between ADHD and tic severity is inconsistent. Some evidence indicates that greater ADHD symptoms are associated with perceived tic worsening, particularly under stressful circumstances, whereas other studies find no significant difference in objective tic severity between children with and without ADHD.

Second, the relationship between ADHD and functional burden appears more consistent. ADHD symptoms are associated with difficulties in quality of life, behavioral regulation, and clinical management. Importantly, these outcomes cannot be reduced to tic severity alone.

Third, the treatment literature does not support the assumption that ADHD treatment inevitably worsens tics. Both pharmacological and behavioral interventions can be useful, although evidence quality varies and treatment should be individualized.

Overall, the evidence is most consistent with a multidimensional model in which ADHD and TS share some developmental vulnerability and interact at the symptom and functional levels. ADHD may amplify clinical burden without necessarily producing more severe tics.

10. Future Directions

Future research should prioritize longitudinal designs that can clarify temporal relationships between ADHD and tic symptoms. Cross-sectional correlations cannot determine whether ADHD symptoms precede tic worsening, whether worsening tics increase attentional difficulties, or whether both are influenced by a third factor.

Studies should also distinguish several outcomes that are often combined under the broad term "severity." These include objective tic severity, perceived tic severity, quality of life, academic functioning, social functioning, emotional regulation, and healthcare utilization.

Large longitudinal cohorts could further investigate whether ADHD symptom trajectories predict changes in tic severity or functional impairment across development.

Multimodal studies combining behavioral assessment with electrophysiology, neuroimaging, and genetic information may also help identify mechanisms of shared vulnerability. Symptom-network methods could be particularly useful for examining whether specific ADHD symptoms, rather than ADHD as a unitary construct, are most strongly connected with specific tic or behavioral symptoms.

Finally, future treatment studies should examine outcomes beyond tic reduction. Functional recovery, quality of life, academic performance, emotional regulation, and caregiver burden may provide a more clinically meaningful assessment of treatment effectiveness.

11. Limitations

This review has several limitations. First, it was designed as a focused narrative review rather than a systematic review or meta-analysis. Consequently, it does not provide a formal risk-of-bias assessment or quantitative synthesis across all available studies.

Second, the included literature varies considerably in sample size, age range, study design, outcome measurement, and clinical context. Some evidence comes from small cross-sectional studies, while other findings are derived from randomized controlled trials, network analyses, longitudinal research, or preclinical models.

Third, some mechanistic evidence comes from populations or models that cannot be directly generalized to children or adolescents with TS and ADHD. Such evidence was therefore used to provide explanatory context rather than direct clinical conclusions.

Fourth, the original narrative screening process was not prospectively logged, meaning that an exact number of records identified, screened, and excluded could not be reconstructed reliably. This limitation prevents the review from making systematic-review-level claims about comprehensiveness.

Finally, the available evidence does not consistently separate the independent effects of ADHD from those of obsessive-compulsive symptoms, anxiety, depression, emotional dysregulation, and environmental stress. Future research should address these overlapping contributors using adequately powered longitudinal designs.

12. Conclusion

ADHD is an important component of the clinical presentation of Tourette syndrome, but its role should not be reduced to a simple association with greater tic severity. Current evidence suggests that ADHD symptoms can interact with tic symptoms within a broader symptom network, while their relationship with objective tic severity remains inconsistent.

The evidence for functional burden is more compelling. ADHD symptoms may contribute to poorer quality of life, behavioral dysregulation, treatment needs, and everyday impairment even when tic severity itself is not substantially elevated. This distinction has important clinical implications because effective management of TS should address the symptoms that most interfere with functioning rather than focusing exclusively on tic reduction.

Current treatment evidence also provides reassurance that ADHD can be treated in individuals with TS without an inevitable worsening of tics. Pharmacological and behavioral interventions should therefore be selected according to individual clinical needs and monitored over time.

Overall, ADHD comorbidity in TS is best understood as part of a multidimensional clinical system involving shared vulnerability, interacting symptoms, and functional consequences. Further longitudinal and multimodal research is needed to clarify the mechanisms underlying this relationship and to determine which symptom domains most strongly predict long-term outcomes.

Table 1.

Selected evidence concerning ADHD comorbidity in Tourette syndrome

Role in review	Key finding	Domain	Design and sample	Study
Direct evidence distinguishing functional burden from tic severity	Tic severity was not significantly associated with QOL, whereas ADHD and OCD symptoms were associated with poorer QOL; inattentiveness predicted lower QOL	Quality of life	Clinical study; $N = 56$ children with GTS	Bernard et al. (2009)
Counterpoint to a simple ADHD–tic severity model	No significant difference in YGTSS severity between children with and without ADHD; ADHD group was more likely to receive tic treatment	Tic severity/treatment	Clinical study; $N = 114$ children	Pringsheim (2017)
Supports CBIT despite ADHD comorbidity	ADHD, OCD, and anxiety did not significantly moderate response to behavioral therapy for tics	CBIT	Behavioral-treatment analysis; combined $N = 248$	Sukhodolsky et al. (2017)
Evidence for context-dependent ADHD–tic association	ADHD symptom severity correlated with perceived tic worsening during the COVID-19	Tic worsening	Cross-sectional clinical study; $N = 22$	Araújo et al. (2022)

	pandemic ($r = .602$, $p = .003$)			
Illustrative clinical evidence; limited generalizability	Described TS, OCD, and ADHD occurring together in a patient with NF1	Clinical co-occurrence	Case report	Kaur et al. (2022)
Evidence for shared biological vulnerability	Identified predominantly male-associated rare X-linked risk across TS, ADHD, and autism	Genetics	Large genetic analysis	Wang et al. (2023)
Supports an interconnected symptom-system model	Identified substantial cross-domain connections between ADHD and TS symptoms; 83 of 360 possible cross-domain edges were non-zero	Symptom interaction	Network analysis; $N = 3,958$ children/adolescents	Zhang et al. (2024)
Treatment evidence	$\alpha 2$ agonists and combined stimulant- $\alpha 2$ treatment improved ADHD and tic outcomes; stimulants improved ADHD without significant tic worsening	Pharmacological treatment	Systematic review and network meta-analysis; 8 RCTs, $N = 575$	Farhat et al. (2024)
Supports behavioral treatment despite ADHD symptoms	ADHD symptomatology did not significantly alter response to CBT for tics	Behavioral treatment	CBT study; $N = 55$	Mazur-Lainé et al. (2024)
Preclinical mechanistic evidence	Postnatal CB1/CB2 disruption produced TS/ADHD-like behavioral features	Mechanisms	Preclinical mouse study	Gorberg et al. (2025)
Healthcare-delivery context; not direct functional-impairment evidence	Examined telehealth and quality of care for individuals with ADHD and TS in Medicaid	Healthcare access	Healthcare-delivery study	Karacuschansky et al. (2025)
Mechanistic/background evidence	Identified altered performance-monitoring markers across OCD, GTS, ADHD, and	Electrophysiology	Systematic review/meta-analysis; 97 studies, 5,890 participants	Bellato et al. (2021)

	autism			
Evidence linking ADHD to behavioral burden	Higher ADHD symptoms were associated with rage attacks and predicted rage attacks at follow-up	Behavioral dysregulation	Longitudinal/cross-sectional study; $N = 314$ at baseline	Cintin et al. (2026)

Note. GTS = Gilles de la Tourette syndrome; ADHD = attention-deficit/hyperactivity disorder; QOL = quality of life; OCD = obsessive-compulsive disorder; YGTSS = Yale Global Tic Severity Scale; CBIT = Comprehensive Behavioral Intervention for Tics; CBT = cognitive-behavioral therapy; RCT = randomized controlled trial.

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